

## JOURNAL CLUB

## Rhinovirus and risk of childhood asthma

Caliskan *et al*<sup>1</sup> report a significant interaction between genotype variants at the 17q21 locus and human rhinovirus (HRV) wheezing illness in early life leading to the development of childhood onset asthma. The authors studied the Childhood Origins of Asthma (COAST) and the Copenhagen Prospective Study on Asthma in Childhood (COPSAC) birth cohorts, as well as 100 adults. Genotype-specific effects of HRV stimulation on peripheral blood mononuclear cells (PBMCs) were examined.

Five asthma-associated 17q21 single nucleotide polymorphisms (SNPs) were genotyped in the COAST and COPSAC cohorts. Each was tested for an association with asthma and phenotypes of viral wheezing illness using either a linear or binary regression model. PBMCs were analysed in vitro from each of the adult participants using PCR to assess the expression of 17q21 polymorphisms in HRV-stimulated and HRV-unstimulated cells. All SNPs examined in the COAST cohort showed significant associations with HRV wheezing illness and the number of illnesses experienced in the first 3 years of life. This association was not found with respiratory syncytial virus (RSV) wheezing illness. Children with the 17q21 genotype and HRV wheezing illness were at increased risk of developing childhood asthma (OR 5.2). This finding was replicated in the COPSAC cohort (OR 3.9).

The authors found significant upregulation of ORMDL3, hypothesised to prevent apoptosis in virally infected cells, in HRV-exposed PBMCs with the 17q21 genotype. They postulated that overexpression of this protein may increase the efficiency of viral infection and prevent HRV clearance.

This study does not provide any evidence of causality and the importance of the aetiological influence of the lung's bacterial microbiome is not examined. It was also underpowered to effectively assess the interaction with RSV-induced wheezing illness. Further research is needed in this area; however, this study highlights the importance of understanding the interaction between genetics and environmental risk factors in the development of airways disease.

**Chris Course**

**Correspondence to** Dr Chris Course, FY2, Department of General Medicine, Llandough Hospital, Penlan Road, Cardiff CF64 2XX, UK; [chriscourse@doctors.org.uk](mailto:chriscourse@doctors.org.uk)

**Competing interests** None.

**Provenance and peer review** Not commissioned; internally peer reviewed.

**To cite** Course C. *Thorax* 2014;**69**:199.

*Thorax* 2014;**69**:199. doi:10.1136/thoraxjnl-2013-204083

**REFERENCE**

- 1 Caliskan M, Bochkov YA, Kreiner-Møller E, *et al*. Rhinovirus wheezing illness and genetic risk of childhood-onset asthma. *N Engl J Med* 2013;**368**:1398–406.